

HHS Public Access

Author manuscript

Lung. Author manuscript; available in PMC 2015 October 01.

Published in final edited form as:

Lung. 2014 October; 192(5): 693-700. doi:10.1007/s00408-014-9609-2.

Active Asthma and the Prevalence of Physician-Diagnosed COPD

Maria C. Mirabelli,

Air Pollution and Respiratory Health Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, 4770 Buford Highway NE, Mailstop F–60, Atlanta, GA 30341, USA

Suzanne F. Beavers, and

Air Pollution and Respiratory Health Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, 4770 Buford Highway NE, Mailstop F–60, Atlanta, GA 30341, USA

Arjun B. Chatterjee

Section on Pulmonary, Critical Care, Allergy, and Immunologic Diseases, Department of Internal Medicine, Wake Forest School of Medicine, Winston-Salem, NC, USA

Maria C. Mirabelli: zif7@cdc.gov

Abstract

Introduction—Despite the considerable overlap of asthma and chronic obstructive pulmonary disease (COPD), the extent to which the two diagnoses are the manifestations of the same disease remains unresolved. We conducted these analyses to evaluate the role of active asthma in the prevalence of physician-diagnosed COPD.

Methods—From 2006 through 2010, 74,209 adults aged 18–99 years and with a history of asthma participated in the Behavioral Risk Factor Surveillance System (BRFSS) Asthma Callback Survey and responded to interview-administered questionnaires via telephone. We used publicly available data from 71,639 (97%) participants to identify respondents with and without active manifestations of asthma and self-reported, physician-diagnosed COPD. We generated population-weighted estimates of physician-diagnosed COPD prevalence and conducted linear regression to estimate associations between active asthma status and the prevalence of COPD among current smokers, former smokers, and lifetime nonsmokers separately.

Results—Physician-diagnosed COPD was reported in an estimated 29% of the population with any history of asthma, including both active and inactive asthma. Age-specific prevalences of physician-diagnosed COPD were consistently higher among adults with active asthma than adults without active asthma. Compared to inactive asthma, active asthma was associated with an 8.3% [95 % confidence interval (CI) 6.1, 10.5] higher prevalence of physician-diagnosed COPD among

Correspondence to: Maria C. Mirabelli, zif7@cdc.gov.

lifetime nonsmokers, a 20.6% (95 % CI 18.0, 23.3) higher prevalence among former smokers, and a 26.7% (95 % CI 22.5, 30.9) higher prevalence among current smokers.

Conclusions—Among adults with a history of asthma, active manifestations of asthma may play an important role in the epidemiology of COPD.

Keywords

Adult; Asthma; COPD; Epidemiology; Respiratory health; Surveillance

Introduction

Asthma and chronic obstructive pulmonary disease (COPD) are common respiratory health conditions. Recent estimates in the United States indicate that in 2010, 11% of children 5–14 years of age, 9% of individuals 15–34 years of age, and 8% of adults aged 35 and older experienced current asthma [1]. The same year, nearly 6% of adults aged 25 years and older reported physician-diagnosed COPD, with the percentages increasing to 10% among adults 65 years and older [2]. The co-occurrence of asthma and COPD diagnoses, often referred to as 'overlap syndrome,' is well described, especially at older ages [3–8].

Despite the substantial overlap, the extent to which the two diagnoses are manifestations of the same disease or distinct entities remains unresolved [4, 6, 7, 9–15]. Asthma affects individuals at younger ages, whereas COPD occurs at older ages [1, 2]. Asthma is associated with eosinophilic inflammation, whereas COPD is associated with neutrophilic inflammation [15]. Common risk factors for both asthma and COPD include outdoor air pollution and occupational exposures [16–19], though cigarette smoking continues to be the most well-described, modifiable risk factor for COPD [20]. A history of asthma itself has been associated with the increased risk of COPD [21] and accelerated lung function decline [22, 23]. In a 20-year follow-up study by Silva et al. [21], individuals with active asthma were found to be at increased risk of developing symptoms of chronic bronchitis, being diagnosed with emphysema, and fulfilling criteria consistent with COPD than were individuals with inactive asthma or no asthma history, even after adjustment for age, sex, and smoking history. The fact that individuals with active asthma are at increased risk of developing COPD relative to individuals with inactive asthma suggests that asthma control may affect the occurrence of COPD.

Improving our understanding of the burden of COPD among adults diagnosed with asthma may provide insight into the role of asthma control in the observed overlap between asthma and COPD. We conducted these analyses to examine the prevalence of physician-diagnosed COPD among adults with a history of asthma using data collected routinely for surveillance of asthma in the United States. We report age-specific prevalences of COPD among adult respondents in the Behavioral Risk Factor Surveillance System (BRFSS) Asthma Call-back Survey with and without active asthma and across categories of cigarette smoking history.

Methods

Asthma Call-back Survey

We conducted these analyses using data from the 2006–2010 BRFSS adult Asthma Callback Surveys. The BRFSS is a state-based, random-digit-dial telephone survey of men and women aged 18 years and older conducted annually in the United States [24]. The Asthma Call-back Survey is a follow-up telephone survey conducted approximately two weeks after the BRFSS among respondents who indicated that they have ever had asthma by responding "yes" to the following question: "have you ever been told by a doctor, nurse, or other health professional that you had asthma?" The BRFSS Asthma Call-back Survey is exempt from Institutional Review Board (IRB) review at the Centers for Disease Control and Prevention; state-specific IRB requirements apply to each of the participating states, the District of Columbia, and Puerto Rico.

Study Sample

We pooled the data collected from BFRSS Asthma Call-back Surveys conducted in 2006, 2007, 2008, 2009, and 2010. The pooled sample included data from 42 geographic areas of the United States (40 states, the District of Columbia, and Puerto Rico). In the participating areas included in our analysis, the Council of American Survey and Research Organization response rates ranged from 41 to 71% in 2006, 36 to 72% in 2007, 35 to 68% in 2008, 36 to 66% in 2009, and 31 to 67% in 2010 [25–27]. Because the Asthma Call-back Survey was not conducted in each geographic area during all 5 years, this analysis includes 22 geographic areas for which 5 years of data were available (Arizona, California, Connecticut, District of Columbia, Georgia, Hawaii, Indiana, Iowa, Kansas, Maine, Maryland, Massachusetts, Michigan, Montana, Nebraska, New Hampshire, New York, Oregon, Texas, Vermont, Washington, and Wisconsin), 9 states for which 4 years of data were available (Florida, Illinois, Missouri, Nevada, New Mexico, Ohio, Oklahoma, Utah, and West Virginia), 3 states for which 3 years of data were available (New Jersey, North Dakota, and Rhode Island), 6 geographic areas for which 2 years of data were available (Alaska, Colorado, Louisiana, Pennsylvania, Virginia, and Puerto Rico), and 2 states for which 1 year of data was available (Alabama and Mississippi). Pooling these data resulted in 74,209 respondents; we then excluded respondents who did not provide information about physician-diagnosed COPD status, cigarette smoking status, age, educational attainment, race/ethnicity, or sex, resulting in a sample of 71,639 respondents (Fig. 1).

Physician-Diagnosed COPD

We adhered to existing definitions to identify respondents with self-reported, physician-diagnosed COPD status [2, 28–31]. Briefly described, the respondents were identified as having self-reported, physician-diagnosed COPD if responses were positive to any of the following three questionnaire items: "Have you ever been told by a doctor or health professional that you have chronic obstructive lung disease, also known as COPD?"; "Have you ever been told by a doctor or other health professional that you have emphysema?"; and "Have you ever been told by a doctor or other health professional that you have chronic bronchitis?" Self-reported, physician-diagnosed COPD is hereafter referred to as "physician-diagnosed COPD."

Identification of Active Asthma Among Adults with a History of Asthma

As described above, each Asthma Call-back Survey respondent was selected because of a positive response during BRFSS data collection about having been told by a doctor, nurse, or other health professional that he/she had asthma; thus, all respondents are considered to have a history of physician-diagnosed asthma. As in previous analysis of Asthma Call-back Survey data [32, 33], we categorized respondents as having *active* asthma if they reported that at least one of the following occurred during the past 12 months: talked to a doctor or other health professional about [his/her] asthma, took asthma medication, or experienced any symptoms of asthma. The remaining respondents, those without active asthma, are hereafter referred to as having *inactive* asthma. Following our main analysis, we conducted a secondary analysis in which we further restricted the categorization of active asthma to include only those respondents who reported experiencing any symptoms of asthma during the past 12 months.

Other Covariates

Each respondent self-reported his/her age, race/ethnicity, sex, educational attainment, and cigarette smoking status. Each respondent's cigarette smoking status was categorized as current smoker, former smoker, or lifetime nonsmoker.

Statistical Analysis

Before pooling data from 2006–2010, we evaluated variation in the estimated prevalences of the variables of interest across the five survey-year period using the Rao-Scott Chi-square test, a design-adjusted variation of the goodness-of-fit test for equal proportions [34]. The resulting test statistics for active asthma ($X^2 = 3.80$, 4 degrees of freedom [df], p = 0.43), physician-diagnosed COPD ($X^2 = 7.66$, 4 df, p = 0.11), current smoking ($X^2 = 1.42$, 4 df, p = 0.84), former smoking ($X^2 = 6.28$, 4 df, p = 0.18), and lifetime nonsmoking ($X^2 = 7.28$, 4 df, p = 0.12) did not indicate differences at $\alpha = 0.05$ in the proportion of respondents in each category across the 5-year period; therefore, we pooled the 5 datasets and present characteristics of the pooled 2005–2010 samples and the weighted population estimates.

To generate weighted population estimates, we used adjusted sampling weights that account for BRFSS and Asthma Call-back Survey nonresponse and unequal sampling probabilities. Unadjusted annual sampling weights were provided with the Asthma Call-back Survey data. Because we pooled data collected from 2006 through 2010 and because the number of geographic areas with Asthma Call-back Survey varied from year to year, we adjusted the sampling weights in each geographic area by dividing the unadjusted weight by the number of years for which data were available from that geographic area. We estimated prevalence of physician-diagnosed COPD within categories of age and across strata of active asthma and smoking status. We used linear regression to estimate associations between active asthma status and the prevalence of physician-diagnosed COPD. For this analysis, a single linear regression model was adjusted for age as a continuous variable centered at 44 years, educational attainment, race/ethnicity, sex, smoking status, and an interaction of active asthma status and smoking status. Results are presented as prevalence differences (PD), with 95% confidence intervals (CI), indicating estimated changes in the prevalence of physician-diagnosed COPD on the absolute scale. Estimated changes in prevalence between the

populations with active asthma and inactive asthma are shown separately for current smokers, former smokers, and lifetime nonsmokers. All analyses were conducted using procedures for analysis of complex sample survey data in SAS version 9.3 (SAS Institute Inc., Cary, North Carolina, USA). Results based on fewer than 50 unweighted respondents (i.e., percentages based upon a denominator <50) or for which the relative standard error is greater than 30% are not reported or interpreted.

Results

Table 1 shows characteristics of the 71,639 Asthma Call-back Survey respondents, estimated to represent nearly 26.4 million adults with a history of asthma, including active and inactive asthma. In this population, an estimated 72% were identified as having active asthma and an estimated 29% were identified as having physician-diagnosed COPD. Among those with active asthma, the estimated prevalence of physician-diagnosed COPD was 34.6% (95% CI: 33.7, 35.6); among the remaining 28% with inactive asthma, the estimated prevalence of physician-diagnosed COPD was 14.7% (95% CI: 13.4, 15.9). Among respondents with active and inactive asthma, unadjusted percentage of respondents with physician-diagnosed COPD increased across categories of age; age-specific percentages were consistently highest among current smokers and lowest among lifetime nonsmokers (Fig. 2).

Adjusted associations between respondent characteristics, including active compared to inactive asthma status, and the prevalence of self-reported, physician-diagnosed COPD are shown in Table 2. Adjusted for age, educational attainment, race/ethnicity, and sex, active asthma was associated with a 26.7% (95% CI: 22.5, 30.9) higher prevalence of physician-diagnosed COPD among current smokers, a 20.6% (95% CI: 18.0, 23.3) higher prevalence among former smokers, and an 8.3% (95% CI: 6.1, 10.5) higher prevalence among lifetime nonsmokers. In this adjusted model, the prevalence of physician-diagnosed COPD was 4.6% (95% CI: 3.0, 6.2) higher among female respondents than among male respondents and PDs decreased monotonically with increasing educational attainment (Table 2).

When we restricted our categorization of active asthma to include only those respondents who reported experiencing any symptoms of asthma during the past 12 months, we excluded 5,769 respondents who were previously categorized as having active asthma, but who only reported talking to a doctor or other health care professional about his/her asthma or taking asthma medication during the past 12 months. Following this restriction, relative to inactive asthma, active asthma was associated with a 27.6% (95% CI: 23.3, 31.9) higher prevalence of physician-diagnosed asthma among current smokers, a 21.9% (95% CI: 19.2, 24.7) higher prevalence among former smokers, and an 8.8% (95% CI: 6.7, 11.0) higher prevalence among lifetime nonsmokers (not shown).

Discussion

We analyzed data from the 2006–2010 Asthma Call-back Surveys and found higher prevalences of physician-diagnosed COPD among adults with active asthma than among adults with inactive asthma. These higher prevalences were observed among current and

former smokers as well as lifetime nonsmokers, suggesting an important role for active asthma in the epidemiology of COPD in each category of smoking status.

Our findings complement recent estimates of COPD prevalence (e.g., age-adjusted prevalence: 6.5%; unadjusted prevalence: 6.8%) [2] and extend evidence from prospective, observational studies suggesting that knowledge of asthma history may provide information about the risk of COPD. Specifically, greater annual declines in FEV₁ have been observed among individuals with self-reported asthma than among those without self-reported asthma, including among both smokers and nonsmokers [22]; greater age-related declines in FEV1 have been reported among participants with asthma and among cigarette smokers [23]; and elevated risks of COPD have been reported among adults with active asthma and among current smokers [21]. Our findings build on this evidence by using public health surveillance data that are collected annually to examine whether the relationship between asthma and physician-diagnosed COPD can be observed in a large, cross-sectional population-based sample. The use of public health surveillance data has been identified as a priority for advancing the prevention and control of COPD [35]. Analysis of such data sources may provide useful information about modifiable factors (e.g., level of asthma control and smoking status), non-modifiable factors (e.g., age and sex), and other characteristics (e.g., educational attainment) associated with the prevalence of physician-diagnosed COPD.

In these analyses, we identified respondents as having active asthma based on positive responses about having talked to a doctor or other health professional about [his/her] asthma (estimated prevalence: 51%), taken asthma medication (estimated prevalence: 58%), and experienced any symptoms of asthma (estimated prevalence: 64%) during the past 12 months. When we restricted our categorization of active asthma to include only those respondents who reported experiencing any symptoms of asthma during the past 12 months, the associations between active asthma and the prevalence of COPD were approximately 1% higher than those observed in our main analysis, where the definition of active asthma included talking to a doctor or other health care professional about asthma or taking asthma medication during the past 12 months. The small change following this restriction suggests that the associations observed in all three categories of smoking status are only modestly attenuated by the outcomes of discussions with doctors or other health care providers or asthma medication use during the past 12 months. In these data, just 4% were categorized as having active asthma by only having talked to a doctor or other health professional about asthma during the past 12 months and just 1% were categorized by only having taken asthma medication during the past 12 months. Using these public health surveillance data, we were unable to identify whether symptoms reported during the past 12 months improved following discussions with doctors or health professionals or use of medications for asthma, among individuals who reported these factors. Additional analyses conducted among populations responding to similar questions at intervals longer than 12 months apart may provide useful data about the contributing roles of health care visits and asthma medications in defining active asthma. Similarly, additional analyses conducted in the Asthma Call-back Survey or other surveillance data may further improve our understanding of how changes in medications and treatment practices, advances in patient education and self-management methods, and trends in second-hand smoke exposures and cigarette smoking may affect the relationship between asthma and COPD.

We analyzed cross-sectional data and do not have in-depth information about factors that may be involved in the development of asthma, COPD, or both. Indeed, because we analyzed surveillance data, we cannot draw conclusions about or contribute to the discussion of whether there exists a causal relationship between asthma and COPD. A major limitation of these data is the lack of information about the extent to which asthma or COPD may have been incorrectly diagnosed or reported; neither the BRFSS nor the Asthma Call-back Survey include means by which the responses may be validated. A comparison of self-reported asthma to asthma classification based on physiologic measures during a clinical exam 10 years earlier has shown that the validity of self-reported asthma varies with asthma severity [36, 37]. In the absence of clinical measures of asthma, participants in survey research are often asked whether they have been told by a doctor, nurse, or other health care provider that they have asthma. Data collected via self-administered questionnaire can be a reliable source of information about asthma [38]; the extent to which validity and reliability of asthma history information collected via interviewer-administered telephone survey are different from validity and reliability of the same information collected via self-administered questionnaire is unknown. Comparable data evaluating the validity and reliability of physician-reported COPD or the magnitude of recall bias affecting self-report of physiciandiagnosed COPD are also unavailable. We reiterate that in these data, self-reported, physician-diagnosed COPD status is considered a proxy for actual disease status. Similarly, self-reported smoking status is considered a proxy for actual smoking status and additional description of current and former smoking habits, such as pack-years of cigarettes smoked, is not available in these data. Information about self-reported smoking status cannot be used to make inferences about the extent to which the prevalence of physician-diagnosed COPD varies within groups, such as among occasional smokers, smokeless-tobacco users, individuals exposed to secondhand tobacco smoke, and those who quit smoking recently versus those who quit long ago, or across categories of pack-years of smoking history.

The large size of the pooled annual survey samples allowed us to evaluate the prevalence of physician-diagnosed COPD within strata of smoking status. We examined an alternate statistical method to adjust sampling weights; revising our analyses with alternate weights, computed by multiplying the proportion of respondents in each survey year by the corresponding survey year's weight, as in previous analyses of Asthma Call-back Survey data [39, 40], did not markedly affect our results or conclusions (data not shown). Pooling data collected over five years provided a sufficiently large sample to enable us to evaluate the prevalence of physician-diagnosed COPD in nonsmoking and young adults, where the prevalences of COPD are relatively low, and in the sparsely populated category of current smokers with inactive asthma. Improving our understanding of the influence of active manifestations of asthma, including recent asthma symptoms, on long-term respiratory health may improve our growing understanding of the epidemiology of COPD. Indeed, cigarette smoking continues to be the most well-described, modifiable risk factor for COPD [20], and smoking prevention efforts continue to be identified as the highest priorities to reduce the onset of new COPD and exacerbation of existing COPD [41]. These findings highlight the importance of appropriate management of asthma among smokers and nonsmokers alike.

Acknowledgments

The Asthma Call-back Survey is jointly administered with the Office of Surveillance, Epidemiology and Laboratory Services, Division of Behavioral Surveillance; data collection is managed by BRFSS coordinators in each of the participating states, the District of Columbia, and Puerto Rico.

References

- Moorman JE, Akinbami LJ, Bailey CM, Zahran HS, King ME, Johnson CA, Liu X. National surveillance of asthma: United States, 2001–2010. National Center for Health Statistics. Vital Health Stat. 2012; 3(35):1–67.
- Ford ES, Croft JB, Mannino DM, Wheaton AG, Zhang X, Giles WH. COPD Surveillance-United States, 1999–2011. Chest. 2013; 144(1):284–305.10.1378/chest.13-0809 [PubMed: 23619732]
- Chang J, Mosenifar Z. Differentiating COPD from asthma in clinical practice. J Intensive Care Med. 2007; 22(5):300–309.10.1177/0885066607304445 [PubMed: 17895488]
- Zeki AA, Schivo M, Chan A, Albertson TE, Louie S. The asthma-COPD overlap syndrome: a common clinical problem in the elderly. J Allergy (Cairo). 2011; 2011:861926.10.1155/2011/861926 [PubMed: 22121384]
- Gibson PG, Simpson JL. The overlap syndrome of asthma and COPD: what are its features and how important is it? Thorax. 2009; 64(8):728–735.10.1136/thx.2008.108027 [PubMed: 19638566]
- Mauad T, Dolhnikoff M. Pathologic similarities and differences between asthma and chronic obstructive pulmonary disease. Curr Opin Pulm Med. 2008; 14(1):31–38.10.1097/MCP. 0b013e3282f19846 [PubMed: 18043273]
- Nakawah MO, Hawkins C, Barbandi F. Asthma, chronic obstructive pulmonary disease (COPD), and the overlap syndrome. J Am Board Fam Med. 2013; 26(4):470–477.10.3122/jabfm. 2013.04.120256 [PubMed: 23833163]
- 8. de Marco R, Pesce G, Marcon A, Accordini S, Antonicelli L, Bugiani M, Casali L, Ferrari M, Nicolini G, Panico MG, Pirina P, Zanolin ME, Cerveri I, Verlato G. The coexistence of asthma and chronic obstructive pulmonary disease (COPD): prevalence and risk factors in young, middle-aged and elderly people from the general population. PLoS One. 2013; 8(5):e62985.10.1371/journal.pone.0062985 [PubMed: 23675448]
- 9. Miravitlles M, Soriano JB, Ancochea J, Munoz L, Duran-Tauleria E, Sanchez G, Sobradillo V, Garcia-Rio F. Characterisation of the overlap COPD-asthma phenotype. Focus on physical activity and health status. Respir Med. 2013; 107(7):1053–1060.10.1016/j.rmed.2013.03.007 [PubMed: 23597591]
- 10. Gibson PG, Simpson JL. The overlap syndrome of asthma and COPD: what are its features and how important is it? Thorax. 2009; 64(8):728–735.10.1136/thx.2008.108027 [PubMed: 19638566]
- Fens N, Zwinderman AH, van der Schee MP, de Nijs SB, Dijkers E, Roldaan AC, Cheung D, Bel EH, Sterk PJ. Exhaled breath profiling enables discrimination of chronic obstructive pulmonary disease and asthma. Am J Respir Crit Care Med. 2009; 180(11):1076–1082.10.1164/rccm. 200906-0939OC [PubMed: 19713445]
- 12. Kraft M. Asthma and chronic obstructive pulmonary disease exhibit common origins in any country! Am J Respir Crit Care Med. 2006; 174(3):238–240.10.1164/rccm.2604007 [PubMed: 16864716]
- Barnes PJ. Against the Dutch hypothesis: asthma and chronic obstructive pulmonary disease are distinct diseases. Am J Respir Crit Care Med. 2006; 174(3):240–243.10.1164/rccm.2604008
 [PubMed: 16864717]
- 14. Bleecker ER. Similarities and differences in asthma and COPD. The Dutch hypothesis. Chest. 2004; 126(2 Suppl):93S–95S.10.1378/chest.126.2_suppl_1.93S [PubMed: 15302768]
- 15. Barnes PJ. Mechanisms in COPD: differences from asthma. Chest. 2000; 117(2 Suppl):10S-14S. [PubMed: 10673467]
- 16. Hnizdo E, Sullivan PA, Bang KM, Wagner G. Association between chronic obstructive pulmonary disease and employment by industry and occupation in the US population: a study of data from the

- Third National Health and Nutrition Examination Survey. Am J Epidemiol. 2002; 156(8):738–746. [PubMed: 12370162]
- 17. Kogevinas M, Zock JP, Jarvis D, Kromhout H, Lillienberg L, Plana E, Radon K, Toren K, Alliksoo A, Benke G, Blanc PD, Dahlman-Hoglund A, D'Errico A, Hery M, Kennedy S, Kunzli N, Leynaert B, Mirabelli MC, Muniozguren N, Norback D, Olivieri M, Payo F, Villani S, van Sprundel M, Urrutia I, Wieslander G, Sunyer J, Anto JM. Exposure to substances in the work-place and new-onset asthma: an international prospective population-based study (ECRHS-II). Lancet. 2007; 370(9584):336–341.10.1016/S0140-6736(07)61164-7 [PubMed: 17662882]
- Salam MT, Islam T, Gilliland FD. Recent evidence for adverse effects of residential proximity to traffic sources on asthma. Curr Opin Pulm Med. 2008; 14(1):3–8.10.1097/MCP. 0b013e3282f1987a [PubMed: 18043269]
- 19. Viegi G, Maio S, Pistelli F, Baldacci S, Carrozzi L. Epidemiology of chronic obstructive pulmonary disease: health effects of air pollution. Respirology. 2006; 11(5):523–532.10.1111/j. 1440-1843.2006.00886.x [PubMed: 16916323]
- Global Initiative for Chronic Obstructive Lung Disease (GOLD). [Accessed 11 June 2013] Global Strategy for the Diagnosis, Management, and Prevention of COPD. Available: http:// www.goldcopd.org/
- 21. Silva GE, Sherrill DL, Guerra S, Barbee RA. Asthma as a risk factor for COPD in a longitudinal study. Chest. 2004; 126(1):59–65.10.1378/chest.126.1.59 [PubMed: 15249443]
- Lange P, Parner J, Vestbo J, Schnohr P, Jensen G. A 15-year follow-up study of ventilatory function in adults with asthma. N Engl J Med. 1998; 339(17):1194–1200.10.1056/ NEJM199810223391703 [PubMed: 9780339]
- James AL, Palmer LJ, Kicic E, Maxwell PS, Lagan SE, Ryan GF, Musk AW. Decline in lung function in the Busselton Health Study: the effects of asthma and cigarette smoking. Am J Respir Crit Care Med. 2005; 171(2):109–114.10.1164/rccm.200402-230OC [PubMed: 15486340]
- Centers for Disease Control and Prevention (CDC). Behavioral risk factor surveillance system. http://www.cdc.gov/asthma/survey/brfss.html. Cited 23 Jan 2013
- National Asthma Control Program. 2006–2008 Behavioral Risk Factor Surveillance System Asthma Call-back Survey summary data quality report. http://www.cdc.gov/brfss/acbs/2008/documentation/SDQReportACBS_06-08.rtf. Cited 26 Aug 2013
- National Asthma Control Program. 2009 Behavioral Risk Factor Surveillance System Asthma Call-back Survey summary data quality report. http://www.cdc.gov/brfss/acbs/2009/ documentation/SDQReportACBS_09.rtf. Cited 26 Aug 2013
- National Asthma Control Program. 2010 Behavioral Risk Factor Surveillance System Asthma Call-back Survey summary quality data report. http://www.cdc.gov/brfss/acbs/2010/ documentation/SDQReportACBS_10.rtf. Cited 11 March 2013
- Pleasants RA, Ohar JA, Croft JB, Liu Y, Kraft M, Mannino DM, Donohue JF, Herrick HL.
 Chronic obstructive pulmonary disease and asthma-patient characteristics and health impairment.
 COPD. Epub ahead of print 23 Oct 2013. 10.3109/15412555.2013.840571
- 29. Akinbami LJ, Liu X. Chronic obstructive pulmonary disease among adults aged 18 and over in the United States, 1998–2009. NCHS Data Brief. 2011; 63:1–8. [PubMed: 22142836]
- 30. Mannino DM, Homa DM, Akinbami LJ, Ford ES, Redd SC. Chronic obstructive pulmonary disease surveillance–United States, 1971-2000. MMWR Surveill Summ. 2002; 51(6):1–16.
- 31. Antwi S, Steck SE, Heidari K. Association between prevalence of chronic obstructive pulmonary disease and health-related quality of life, South Carolina, 2011. Prev Chronic Dis. 2013; 10:E215.10.5888/pcd10.130192 [PubMed: 24370110]
- 32. Zahran HS, Person CJ, Bailey C, Moorman JE. Predictors of asthma self-management education among children and adults–2006–2007 Behavioral Risk Factor Surveillance System Asthma Callback Survey. J Asthma. 2012; 49(1):98–106.10.3109/02770903.2011.644012 [PubMed: 22216949]
- 33. Mirabelli MC, Beavers SF, Chatterjee AB, Moorman JE. Age at asthma onset and subsequent asthma outcomes among adults with active asthma. Respir Med. 2013; 107(12):1829–1836.10.1016/j.rmed.2013.09.022 [PubMed: 24139624]

34. Rao JNK, Scott AJ. On chi-squared tests for multiway contingency tables with cell proportions estimated from survey data. Ann Stat. 1984; 12(1):46–60.10.2307/2241033

- 35. Centers for Disease Control and Prevention (CDC). Public health strategic framework for COPD prevention. Atlanta, GA: Centers for Disease Control and Prevention; 2011. http://www.cdc.gov/copd/pdfs/framework_for_copd_prevention.pdf. Cited 16 May 2014
- 36. Balder B, Lindholm NB, Löwhagen O, Palmqvist M, Plaschke P, Tunsäter A, Torén K. Predictors of self-assessed work ability among subjects with recent-onset asthma. Respir Med. 1998; 92(5): 729–734. [PubMed: 9713631]
- 37. Torén K, Palmqvist M, Löwhagen O, Balder B, Tunsäter A. Self-reported asthma was biased in relation to disease severity while reported year of asthma onset was accurate. J Clin Epidemiol. 2006; 59(1):90–93.10.1016/j.jclinepi.2005.03.019 [PubMed: 16360566]
- 38. Mirabelli MC, Beavers SF, Flanders DW, Chatterjee AB. Reliability in reporting asthma history and age at asthma onset. J Asthma. 201410.3109/02770903.2014.930480
- Knoeller GE, Mazurek JM, Moorman JE. Characteristics associated with health care professional diagnosis of work-related asthma among individuals who describe their asthma as being caused or made worse by workplace exposures. J Occup Environ Med. 2012; 54(4):485–490.10.1097/JOM. 0b013e3182479f93 [PubMed: 22453813]
- 40. Knoeller GE, Mazurek JM, Moorman JE. Work-related asthma, financial barriers to asthma care, and adverse asthma outcomes: Asthma Call-back Survey, 37 states and District of Columbia, 2006 to 2008. Med Care. 2011; 49(12):1097–1104.10.1097/MLR.0b013e31823639b9 [PubMed: 22002642]
- 41. de Marco R, Accordini S, Marcon A, Cerveri I, Anto JM, Gislason T, Heinrich J, Janson C, Jarvis D, Kuenzli N, Leynaert B, Sunyer J, Svanes C, Wjst M, Burney P. European Community Respiratory Health S. Risk factors for chronic obstructive pulmonary disease in a European cohort of young adults. Am J Respir Crit Care Med. 2011; 183(7):891–897.10.1164/rccm. 201007-1125OC [PubMed: 20935112]

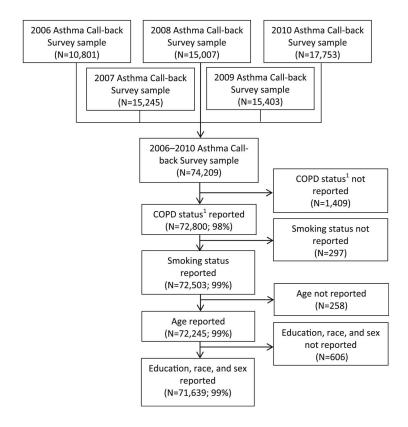


Fig. 1. Selection of the survey respondents: 2006–2010 Asthma Call-back Survey. ¹self-reported, physician-diagnosed COPD

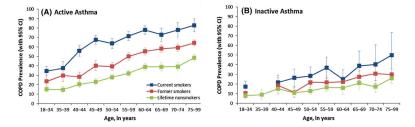


Fig. 2. Age-specific estimated prevalences of physician-diagnosed COPD by smoking history among adults with active asthma (*panel A*) and inactive asthma (*panel B*). Estimates based on fewer than 50 unweighted respondents or for which the relative standard error is greater than 30% are not shown

Author Manuscript

Table 1

Characteristics of the selected survey respondents and weighted population estimates of adults with a history of asthma by active asthma status: 2006– 2010 Asthma Call-back Survey

	Total population			By active asthma	
	Survey respondents No.	Weighte	Weighted population estimate	Yes	No
		No.a	Percentage (95% CI)	Percentage (95% CI) with ${ m COPD}^b \omega$	Percentage (95% CI) with ${ m COPD}^b$, c
Total	71,639	26,408			
Demographic characteristics					
Age					
18–34	9,094	9,122	34.5 (33.5, 35.5)	20.4 (18.1, 22.7)	10.5 (8.2, 12.7)
35–39	4,568	2,311	8.8 (8.3, 9.2)	22.3 (19.5, 25.2)	9.9 (6.1, 13.7)
40-44	6,533	3,107	11.8 (11.2, 12.3)	30.4 (27.8, 33.1)	17.0 (13.1, 20.9)
45-49	5,669	1,840	7.0 (6.6, 7.3)	37.8 (35.1, 40.5)	13.3 (10.2, 16.3)
50–54	8,536	2,600	9.8 (9.4, 10.3)	39.3 (37.1, 41.5)	18.6 (15.3, 22)
55–59	9,406	2,022	7.7 (7.3, 8.0)	45.4 (43.1, 47.7)	20.6 (17.4, 23.9)
60–64	8,589	1,775	6.7 (6.4, 7.0)	52.5 (50.1, 54.8)	19.4 (15.4, 23.5)
65–69	6,789	1,275	4.8 (4.6, 5.1)	51.7 (48.9, 54.5)	25.2 (21.0, 29.5)
70–74	5,102	968	3.4 (3.2, 3.6)	52.6 (49.8, 55.4)	24.9 (20.2, 29.5)
75–99	7,353	1,460	5.5 (5.3, 5.8)	58.2 (55.8, 60.5)	28.7 (24.7, 32.8)
Educational attainment					
Less than high school	6,260	2,459	9.3 (8.8, 9.8)	52.3 (49.1, 55.5)	23.6 (16.9, 30.3)
Graduated high school	18,386	6,554	24.8 (24.0, 25.6)	43.3 (41.1, 45.5)	16.9 (14.5, 19.3)
Some college/technical school	21,179	7,596	28.8 (27.9, 29.6)	35.0 (33.4, 36.7)	14.9 (12.4, 17.3)
Completed college/technical school	25,814	008,6	37.1 (36.3, 38)	23.0 (21.6, 24.3)	11.8 (10.1, 13.6)
Race/ethnicity					
Black, non-Hispanic	4,232	2,385	9.0 (8.5, 9.6)	33.3 (30.1, 36.5)	13.2 (9.0, 17.4)
White, non-Hispanic	58,442	19,202	72.7 (71.8, 73.6)	35.0 (34.0, 36.0)	15.0 (13.6, 16.5)
Other, non-Hispanic	5,041	1,847	7.0 (6.4, 7.6)	39.1 (34.9, 43.4)	10.9 (7.5, 14.3)
Hispanic	3,924	2,974	11.3 (10.6, 12.0)	30.7 (26.6, 34.9)	16.2 (11.9, 20.6)
Sex					
Female	50,386	15,357	58.2 (57.2, 59.1)	37.1 (36.0, 38.2)	17.4 (15.8, 19.0)

	Total population			By active asthma	
	Survey respondents No. Weighted population estimate	Weighte	d population estimate	Yes	No
		No.a	Percentage (95% CI)	Percentage (95% CI) with ${\rm COPD}^{b,c}$ Percentage (95% CI) with ${\rm COPD}^{b,c}$	Percentage (95% CI) with $\mathrm{COPD}^b x$
Male	21,253	11,051	41.8 (40.9, 42.8)	30.5 (28.6, 32.3)	12.4 (10.5, 14.3)
Health-related characteristics					
Active asthma					
No	17,158	7,372	27.9 (27.1, 28.8)	ı	14.7 (13.4, 15.9)
Yes	54,481	19,035	72.1 (71.2, 72.9)	34.6 (33.7, 35.6)	I
${\rm COPD}^{\mathcal{C}}$					
No	45,742	18,731	70.9 (70.1, 71.7)	ı	I
Yes	25,897	7,677	29.1 (28.3, 29.9)	ı	I
Smoking status					
Current smoker	12,546	5,009	19.1 (18.3, 19.7)	53.0 (50.7, 55.2)	20.5 (16.9, 24.1)
Former smoker	23,969	6,895	26.1 (25.4, 26.8)	44.1 (42.5, 45.7)	18.2 (16.2, 20.3)
Lifetime nonsmoker	35,124	14,503	54.3 (54.0, 55.8)	23.4 (22.1, 24.7)	11.3 (9.7, 13.0)

aIn thousands

 b Percentage of the weighted population estimate

^cPhysician-diagnosed COPD

Table 2

Associations between respondent characteristics and the prevalence of self-reported, physician-diagnosed COPD among 2006–2010 Asthma Call-back Survey respondents

Characteristic	PD (95% CI) ^a
Age, per year	0.6 (0.6, 0.7)
Educational attainment	
Less than high school	18.5 (15.5, 21.6)
Graduated high school	13.0 (10.8, 15.2)
Some college/technical school	7.5 (5.8, 9.2)
Completed college/technical school	0.0 (referent)
Race/ethnicity	_b
Sex	
Female	4.6 (3.0, 6.2)
Male	0.0 (referent)
Smoking status	
Current smoker	
Active asthma	26.7 (22.5, 30.9)
Inactive asthma	0.0 (referent)
Former smoker	
Active asthma	20.6 (18.0, 23.3)
Inactive asthma	0.0 (referent)
Lifetime nonsmoker	
Active asthma	8.3 (6.1, 10.5)
Inactive asthma	0.0 (referent)

 $^{^{}a}$ Prevalence differences (with 95% confidence intervals) generated from a single model including all covariates shown in the table

 $[^]b$ Race/ethnicity is included in the model; however, relative standard errors are greater than 30%; therefore, the results are not shown or interpreted